

**INFORMATION DISCLOSURE
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First Named Inventor	Adams, John W.
Art Unit	1646
Examiner Name	Li, Ruixiang
Attorney Docket Number	AREN-027 (27.US2.PCT)

1	ADAMS, et al. G-proteins in growth and apoptosis: lessons from the heart. <i>Oncogene</i> . 2001, vol. 20, no.13, pp. 1626-1634.	<input type="checkbox"/>
2	BIALIK, et al. The mitochondrial apoptotic pathway is activated by serum and glucose deprivation in cardiac myocytes. <i>Circulation Research</i> . 1999, vol. 85, no. 5, pp. 403-414.	<input type="checkbox"/>
3	BONAVITA, et al. H9c2 cardiac myoblasts undergo apoptosis in a model of ischemia consisting of serum deprivation and hypoxia: inhibition by PMA. <i>FEBS Letters</i> . 2003, vol. 536, pp. 85-91.	<input type="checkbox"/>
4	CERBAI, et al. Isolated cardiac cells for electropharmacological studies. <i>Pharmacological Research</i> . 2000, vol. 42, no. 1, pp. 1-8.	<input type="checkbox"/>
5	LONG, et al. Sympathetic modulation of the cardiac myocyte phenotype: studies with a cell-culture model of myocardial hypertrophy. <i>Basic Research in Cardiology</i> . 1992, vol. 87, Suppl 2, pp. 19-31.	<input type="checkbox"/>
6	PARKER, T. Molecular biology of myocardial hypertrophy and failure: gene expression and trophic signaling. <i>New Horizons</i> . 1995, vol. 3, no. 2, pp. 288-300.	<input type="checkbox"/>
7	SCHAUB, et al. Various hypertrophic stimuli induce distinct phenotypes in cardiomyocytes. <i>Journal of Molecular Medicine</i> . 1997, vol. 75, pp. 901-920.	<input type="checkbox"/>

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